

## Editorial Comment

# Exercise Training After Anterior Q Wave Myocardial Infarction: Harmful or Beneficial\*

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Clinical and experimental studies show that left ventricular dilation may occur after acute myocardial infarction because of myocardial thinning and aneurysm formation at the infarct zone, "infarct expansion" and increased radius of the non-infarcted myocardium (1-5). The dilation may occur early or late and may be progressive. Further, the dilation is more likely to be associated with anterior than with inferior or posterior myocardial infarction, possibly because there is a larger mass of muscle necrosis in anterior infarction. Left ventricular dilation may be an independent risk factor in patients with coronary artery disease (6-8).

**Mechanism of infarct dilation.** Neither a history of reinfarction nor recurrent angina pectoris is a prerequisite for dilation. The process of dilation has been referred to as "remodeling of the left ventricle" and was shown by Pfeffer et al. (5) to be attenuated by long-term therapy with an angiotensin-converting enzyme inhibitor; such therapy improved survival in the animal model. The precise mechanism of dilation is not known, but McKay et al. (9) postulated that the remodeling is a response to normalize wall stress resulting from volume overload. The volume overload may be the result of depression of systolic function, impaired diastolic relaxation or early expansion of the infarct zone. The volume overload results in increased diastolic wall stress and, hence, acts as a stimulus to series replication of sarcomeres and eccentric hypertrophy. An increase in wall stress in both the infarct and noninfarct zones has been found at cardiac catheterization (3). It is possible that several factors may modify the remodeling, such as the extent and site of infarction, hypertension, medications, continued ischemia (both perinfarction and remote) and other yet

undefined factors (see discussion to follow). Areas of viable but ischemic myocardium have been observed at rest with radionuclide (thallium-201) and positron emission tomographic techniques (with nitrogen-13 ammonia and fluoro-2-deoxyglucose) (10). It should be noted that, despite left ventricular dilation, the global ejection fraction may improve in some patients many months after acute infarction (3).

**Role of exercise training after myocardial infarction.** Exercise training has been recommended for patients after acute myocardial infarction, as well as for patients with other cardiac conditions, because of its beneficial effects (11-17). It has been shown, for example, that exercise training decreases the heart rate at rest, increases exercise time, exercise work load and maximal oxygen uptake capacity, decreases heart rate and blood pressure at comparable work loads (compared with pretraining measurements), reduces exercise-induced ischemia and high density lipoprotein cholesterol and improves collateral flow and the psychological well-being of patients. Left ventricular dilation has also been observed during training. The beneficial effects have been attributed to peripheral adaptations that result in a larger oxygen extraction by the skeletal muscles. Some studies suggest that central adaptation can also occur as a result of improved myocardial oxygenation and left ventricular function. However, reported improvement in rest or exercise myocardial perfusion or function, or both, has been inconsistent. It is possible that this inconsistency is related to patient selection, inadequacy of methods of assessment or the intensity or duration of training.

Rogers et al. (18) studied the effect of 7 years of intensive exercise training in nine men with coronary artery disease and found that maximal oxygen uptake capacity continued to show improvement over the 7 year period. This is a remarkable observation because a 10% per decade decline in maximal oxygen capacity due to primary aging after the age of 25 years is expected.

**The current study.** In this issue of the journal, Jugdutt et al. (19) report their observations of deterioration in left ventricular function after exercise training in some patients with anterior myocardial infarction. The authors used a detailed two-dimensional echocardiographic analysis of left ventricular asynergy, volume and topographic variables including the degree of regional shape distortion. All of their 46 patients had a first acute anterior Q wave myocardial infarction; 22 were enrolled in an exercise training program for 12 weeks and 24 patients served as a control group. Only 13 of the initial 22 patients completed the training program. In 6 of 13 patients with a large area of initial asynergy ( $\geq 18\%$ ), topographic and functional deterioration occurred after the training. Such deterioration was not seen in the patients with a small area of initial asynergy ( $< 18\%$ ) or in the 24 patients

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who did not undergo exercise training. Importantly, the deterioration was not reversible as the trend persisted up to the final follow-up at 40 months.

These results appear to be different from those of Cobb et al. (17), who found no change in left ventricular ejection fraction at rest in 15 patients enrolled in a 5 month exercise training program at a mean of 15 weeks after acute myocardial infarction. However, only 4 of these 15 patients had an ejection fraction at rest <50% before enrollment, and no detailed topographic measurements were provided. It is possible, therefore, that these 15 patients were comparable with the group studied by Jugdutt et al. (19) that had a small area of asynergy and had no deterioration with exercise training. Although others (14) have shown that exercise training is safe in patients with severe left ventricular dysfunction, it is likely that these patients had remote infarction and therefore are different from the patients of Jugdutt et al., who were studied a mean of 15 weeks after acute infarction.

Is it possible that the results of Jugdutt et al. (19) are due to errors of measurement? This appears unlikely; the authors have a commendable track record in using similar techniques in previous studies (20,21), their results appear to be highly reproducible and the echocardiographic and radionuclide assessment of left ventricular ejection fraction agreed quite well.

Why did six patients have worsening of regional and global left ventricular function during training? The answer is not clear. None of these patients had a recognizable episode of reinfarction. The authors postulated incomplete healing of a large infarct, which predisposes to expansion and further distortion in topography. Other possible factors that need to be considered include the presence and frequency of silent ischemia (all five patients who underwent catheterization had multivessel coronary artery disease) and the functional adequacy of collateral vessels.

**Clinical implications.** The contribution of Jugdutt et al. (19) has important clinical implications. Until further studies become available it may be advisable not to enroll patients with a large anterior Q wave infarction in an exercise training program soon after infarction. Future studies should address the optimal time of recruiting such patients and the role of silent ischemia as well as whether this deterioration can be modified or prevented by medical therapy and whether it is also seen in patients who have had thrombolytic therapy.

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